

diaphragm, with the result that the normal outward movement of the costal margins will be increased.

3. The presence of a retraction or of an abnormal outward flaring of the subcostal angle will often be of aid in the explanation of obscure diseases of the viscera which lie immediately above or immediately below the diaphragm, especially in pericardial effusion or in subphrenic abscess.

PLEURISY.

BY GEORGE S. GERHARD, M.D.,

PHYSICIAN IN CHIEF, BRYN MAWR HOSPITAL.

PLEURISY in one of its many forms is the commonest of the diseases of the chest, and the most unmanageable. It is often less easy of successful treatment than pneumonia, and sometimes leaves behind it more permanent mischief. Slight attacks may occur without being recognized, explaining the frequency of pleuritic adhesions found in the postmortem examinations of persons who were never known to have been ill. It occurs at all ages, from early infancy to advancing years, but is more frequent in the male between youth and middle age. Its etiological data, bacterial or general, are not, in our present knowledge, quite well enough understood to establish a definite classification. The disease is usually one-sided, preferably confined to the right, though occasionally it is bilateral, and then is apt to be secondary to phthisis or septicemia. It is primary or secondary. By the term primary is meant inflammation of the pleura in a time of health, being mainly limited to the membrane itself, with secondary constitutional results of fever, or if exudation be present, to the mechanical effects of pressure in addition. Secondary pleurisies are by far the more common and the more serious, associated as they so often are with disease of the lungs or neighboring viscera, and sometimes complicating infective processes in the body.

Pleurisies are also divided into the acute and chronic, based upon clinical symptoms, often found, however, to be of doubtful significance. The chronic is frequently of that form from the onset, but it is more likely to be consecutive to the acute. The chief varieties of pleurisy are the fibrinous, the serofibrinous, and the purulent. The predominant causative bacteria are the tubercle bacilli, the pneumococci, and the streptococci. The exudates are often found to be sterile and when a purulent exudate is sterile it is invariably tuberculous. Bacilli of lesser importance exist in the mixed forms.

Fibrinous or plastic pleurisy is often confined to a circumscribed area, lasting for a few days and terminating by adhesions. Its

friction sound is not propagated, but dies where it was born and may readily escape detection. Diffused spots of subacute inflammation not uncommonly occur in connection with chronic nephritis, and if there be friction in the cardiac region it may be influenced by the rhythm of the heart as well as by respiration.

Chronic dry pleurisy, which is usually tuberculous, has in many instances an acute onset, causing thickening of the pleura, extensive adhesions, and fibrosis of cortical portions of the lung. It may remain localized for months or even for years, finally proving fatal from pulmonary phthisis or from acute miliary tuberculosis. I have known of a case of extensive plastic pleurisy of many years' duration to terminate fatally from pneumothorax. The autopsy revealed extensive adhesions binding the lung posteriorly from base to apex. The non-adherent portions of the lung were compressed backward by accumulated air from a ruptured tuberculous ulcer. The pleural cavity was practically free from liquid.

Acute fibroserous pleurisy attacking one in apparent good health after exposure to cold or possibly to traumatism begins with a chill or usually a sense of chilliness, stabbing pain in the chest, disturbed breathing, and fever. A pronounced chill occurs less frequently in pleurisy than in pneumonia, algid attacks taking the place of the former at irregular intervals during the first few days. Pain, on the contrary, is usually intense in pleurisy. It is lancinating, as if from the thrust of a sharp instrument, and often makes the patient cry out on taking a deep breath or after a cough. It is also increased by pressure and by movements of the body.

The pain lessens or ceases altogether when adhesions occur, or when the inflamed surfaces of the pleura are separated by liquid effusion, though it may return intermittently during the patient's illness from exacerbations of the original attack. The seat of election of pleuritic pain is a few fingers' breadth below the nipple, though its point of maximum intensity may be elsewhere in the lateral region of the chest or posteriorly below the inferior angle of the scapula. Occasionally the pain is referred to the lower dorsal region or to some part of the abdomen, giving rise, in the absence of careful examination of the chest, to grave errors in diagnosis. I have known of two patients suffering from pain in the abdomen, one a girl of five years, the other a man about forty years, to be operated upon, the one for appendicitis, the other for perforated gastric ulcer. Both cases ended fatally, showing at the autopsy double serofibrinous pleurisy in the child, and localized diaphragmatic pleurisy in the man. The different seats of pain do not throw clear light upon the precise localization of the disease or its extent, but merely indicate the side affected. Sometimes, however, the pain of circumscribed pleurisy corresponds exactly with the seat of inflammation. In double pleurisy, pain is often felt on one side alone or it may be confined to the middle of the thorax. It is rarely itself

double, but may change from one side to the other, according to the progress of the disease, each one of the pleuræ being successively involved.

The respirations at first are always embarrassed and painful in consequence of efforts at full breathing, the inspiration being cut short before it is completed. But there is not true dyspnea until the lung is compressed by effusion, and perhaps not then unless it be rapid and copious. Cough is sometimes wanting, but in the majority of cases the patient has a short, dry, shallow cough with attempts at restraint, because of the pain to which it gives rise. It is occasionally attended with scanty expectoration of thin, frothy mucus, but if it be abundant and opaque, an association with bronchitis or other affection of the lung is found to exist. In recent cases of moderate severity the pulse bears a close relation to the height of the fever, and is not greatly in excess of the normal. In cases of large effusion with sudden compression of the lung there is a disturbed balance between the pulmonary and general circulation, sufficient blood not passing from the right ventricle into the lungs, and through them to the left side of the heart and the arterial system. Under these circumstances the action of the heart is violent and the pulse at the wrist is frequent and repressed to such a degree as to cause danger from suffocation.

A young man under my care, laboring under an acute pleuritic attack with large effusion, left his bed in the absence of the nurse and walked some distance to the end of the room, the exertion proving almost fatal. On his return to bed the patient was gasping for breath, his heart was beating violently, and his pulse was indicative of lowered arterial tension. Venesection was followed by prompt relief to the threatening symptoms.

The fever of acute pleurisy is of remittent type, with exacerbations toward evening, lasting until the exudate reaches its maximum, when it gradually declines without an attempt on the part of nature to promote a crisis. In some cases the fever returns at irregular intervals, but it finally drops to the normal. Should the temperature be persistently elevated and intermittent there is every reason to suspect the existence of pus in the pleural cavity.

The decubitus of the patient suffering from pleurisy is frequently changed during the course of the malady. At the onset, when there is pain and tenderness on pressure, while the patient avoids lying directly on the affected side, his body is often inclined in that direction. Late in the disease he assumes the dorsal decubitus, but when the effusion is very great the patient naturally prefers lying on the diseased side to relieve the healthy lung of the weight of the fluid. The duration of the active symptoms of acute primary pleurisy with moderate effusion is from ten to twenty days, when absorption begins progressing slowly, however, before the process is completed, often lasting for many weeks. Retraction of portions of the

chest, impairment of resonance, and feebleness of breath sounds with coarse friction may exist for an indefinite length of time, the patient being able to walk about, though looking ill and suffering from dyspnea on exertion.

Chronic pleurisy, as a rule, is the continuation of the acute, though it may result from some low-grade infection from the beginning, in which case the greater number are secondary to a constitutional malady or to some visceral disease. The affection sometimes exists in the form of what is termed latent pleurisy, without any of the general symptoms, unless dyspnea be present, and yet one side of the chest is found to be largely filled with fluid. The physical signs alone reveal the disease, and in every case of suspected infection of the chest a careful examination should always be made.

The following case is mentioned to show how a person laboring under pleurisy of chronic form with enormous effusion was able to perform the full duties of his position without much inconvenience:

C. L., a Chinaman by birth, and cook in a private family, aged sixty-six years, was admitted into the Bryn Mawr Hospital on April 1, 1914. He had been troubled with cough, slight pain in the right side, and shortness of breath for about four weeks, but did not give up work until the day of his admission. On examination the right side of his chest was found to be distended, and there was scarcely any perceptible movement during respiration. There was flatness on percussion and absence of vocal vibrations from three inches below the clavicle in front and below the spine of the scapula posteriorly. At the apex of the lung front and back the percussion note was high-pitched and tympanitic. The breath sounds were altogether absent in the dull regions. Above the level of the fluid posteriorly, and near the spinal column over the compressed lung, there was bronchial respiration and bronchophony. The heart was not much displaced. On April 3, the effusion was so great that the whole of the right side of the chest was absolutely dull on percussion, and breath sounds were everywhere absent except at the root of the lung. Aspiration of the chest was performed, with the result of removing 2200 c.c. of serous fluid, afterward found to be sterile. The patient left the hospital April 23 much improved in health, but suffered a relapse and was readmitted on May 3. The right chest had already undergone retraction. There was dullness posteriorly below the angle of the scapula, and the breath sounds were harsh and distant. Elsewhere on the right side there was tympanitic dullness on percussion and an approach to bronchial breathing. On July 3 the patient was discharged for the second time, with the view of being sent to a sanitarium for outdoor treatment.

Two boys under my care were also cases of latent pleurisy, the first, aged ten years, had passed a section of tape-worm shortly before my visit, and I found him in bed suffering from abdominal

pain, naturally attributed to the irritation of the parasite. There was an absence of symptoms referable to the chest except slight dyspnea, which led me to discover that the patient's left pleural cavity was more than half filled with fluid. The second boy, aged five years, was brought to the country to recover from a gastric attack. He had slight fever, epigastric tenderness, nausea, and vomiting. On examining his chest I found a large effusion on the right side, with all of the characteristic physical signs of pleural inflammation. Both of these patients made good recoveries.

A remarkable instance of pleurisy without marked symptoms was that of Mrs. R., aged forty-five years, who when I first saw her was up and about attending to her household affairs. I was informed that she had been indisposed for a week or more, that she had occasional cough but without pain, and was feverish at night. Examination of the chest revealed dulness on percussion, with absence of breath sounds and of vocal fremitus over the whole of the right lung. There was entire absence of movement of the right side of the chest during respiration, the intercostal spaces were bulging, and the adjacent organs displaced. I left the patient's house to arrange for immediate tapping of the chest, and before I had time to return a message was received that sudden death had occurred after the patient's exertion of going up stairs to the second floor.

The modern practice of early thoracentesis has notably lessened sudden death in pleurisy, though it is still occasionally reported. I once knew fatal syncope to occur, at the Bryn Mawr Hospital, during operation for the removal of fluid from the chest of a robust colored man suffering from acute pleurisy. An autopsy was not allowed.

In cases of pleurisy of long standing neglect the chest is sometimes enormously distended and the comparative immobility between the two sides is most remarkable, the patient raising up the sound side while that of the seat of disease is almost quiet. The intercostal spaces are widened, effaced, and sometimes bulging. The nipple is abnormally distant from the central line of the sternum, and I have often observed it elevated. The neighboring viscera are displaced, chief among them being the heart, which is carried far to the opposite side of the chest. After absorption of the fluid or when it has been mechanically removed, atmospheric pressure is found to have pushed in the parietes of the chest toward the unexpanded lung, giving rise to contraction not only in circumference but from above downward, displacing the scapula and obliterating the intercostal spaces by encroachment of the ribs. The shoulder droops and the body inclines to the affected side, giving the patient a peculiar "lob-sided" gait. In a right-sided pleurisy under these circumstances the liver is drawn up and the heart sometimes pulled over to the right, quite the reverse of what happens when effusion exists.

In a chronic pleuritic case coming under earlier observation the lung, though pushed upward toward the mediastinum or spinal fossa, is less adherent and reëxpands more fully when absorption of the fluid takes place, or is withdrawn by aspiration, and therefore retraction of the chest is less marked. Purulent pleurisy occasionally follows injury to the chest with deep-seated lacerations or it is not infrequently secondary to an acute infective disease. Puerperal pleurisy is often purulent, bilateral, and consequently fatal. Primary suppurative pleurisy is extremely rare in the adult. It is almost always serofibrinous at first, becoming purulent during its subsequent course. It is a common disease of infancy and childhood, and in many instances at that period of life it is purulent from the beginning. The general symptoms are not in any particular different from those of serous pleurisy, with the exception of the persistence of fever and its marked evening rise. Before the days of antisepsis there were many errors of diagnosis and a high mortality of empyema, but now exploratory puncture leads to prompt recognition of the disease and immediate operation. Formerly attempts of nature at spontaneous cure were met with by the discharge of pus through a perforated bronchus or by the formation of a subcutaneous abscess in the parietes of the chest communicating with the pleura, *pleuritis necessitatis*.

In 1886 Miss S., aged eighteen years, was under my care, suffering from acute serous pleurisy. On my first visit there was unmistakable evidence of left-sided pleurisy. Effusion took place rapidly and was copious. In the second week two pints of semi-opaque fluid were removed from the pleural cavity by aspiration. Cough was excessively annoying, and was continuously troublesome until the beginning of the fourth week, when after a violent paroxysm the patient expectorated a large quantity of extremely fetid pus. The discharge was brought up after cough, and on changes of position for many weeks. The patient became profoundly hectic and greatly emaciated, but after a prolonged illness recovered. She is now living and in good health.

Another incident of empyema is perforation of the diaphragm and the escape of pus into the peritoneal cavity, causing local or general inflammation. I have known of two such cases, both terminating fatally after operation. One, a young lady, the subject of tuberculous pleurisy of acute onset, with a copious effusion lasting for many weeks before the fluid was appreciably absorbed. In course of time her general health was sufficiently improved to enable her to go to the far west in search of a suitable climate, which was successfully accomplished, with good results. A second attack of pleurisy, however, was soon followed by suppuration, and pus discharged through the diaphragm, causing a walled-off abscess in the right hypochondrium.

My second patient, a saloon-keeper living in the West, aged forty

years, was attacked with right-sided pleurisy, the effusion becoming rapidly purulent, perforating the diaphragm, and causing general peritonitis. The disease was far advanced when the patient came under observation, and being an extreme alcoholic, he developed septicemia and died soon after being operated upon.

An incident of extension of infection from below upward was that of John C., aged fifty years, admitted into the Bryn Mawr Hospital on June 23, 1914, having a subphrenic abscess associated with pyopneumothorax, caused by perforation of the diaphragm, establishing a communication between the subdiaphragmatic region and the right pleura. The symptoms were abdominal and thoracic, the former more especially, as shown by a large, painful area of resistance in the hypochondrium. The thoracic symptoms were high-pitched tympanitic resonance over the lower half of the right chest, restriction of movement during respiration, with muffled bronchial and occasionally amphoric breathing posteriorly below the angle of the scapula. The normal liver dullness was replaced by tympanitic resonance on percussion. Under local anesthesia a free vertical incision was made in the right hypochondrium, giving exit to a large quantity of extremely fetid pus containing gas. A counter-opening was made in the dependent part of the abscess to insure free drainage. Several weeks later the chest was opened in the anterior axillary line and the sixth rib resected. The patient recovered and left the hospital on November 23, 1914.

A rare case of pleuritis acutissima of foudroyant type, rapidly becoming empyemic, was recently under my observation at the Bryn Mawr Hospital. Ross P., aged thirty-four years, a teamster, of intemperate habits, was admitted into the Hospital on December 12, 1914. The history he gave was that he took cold from exposure in a rain storm, and was confined to the house for one week before coming to the hospital with a troublesome cough, extreme malaise, and finally a chill and pain in the left side. He was complaining on admission of sharp pain in the left side and difficulty in breathing. He had a high temperature, frequent pulse, and excessive cough, with mucous blood-stained expectoration. The tongue, dryish and brown, was tremulously protruded. He was incoherently delirious, and was inclined to get out of bed. There was dullness on percussion over the lower half of the left lung posteriorly, with absent vocal fremitus, feeble breath sounds, much prolonged expiration, and distinct egophony below the angle of the scapula. On December 13 the patient became violently delirious and jumped out of the window, falling on the ground, a distance of twelve feet, but without sustaining serious injury. The patient's delirium lasted for a week or more with fever going at one time to 105°, and on December 25, on exploratory puncture, the effusion was found to contain pus. Owing to the patient's excitable condition, operation upon the chest was delayed until January 5, 1915, when 600 c.c.

of purulent fluid were evacuated from the chest by incision and resection of one rib. The infection was found to be pneumococcic. The patient is now, January 14 recovering.

Prominent among other varieties of pleurisy depending for the most part upon anatomical conditions are the diaphragmatic, hemorrhagic, interlobar, and the pleuropneumonic.

In restricted pleurisy when the diaphragm is the exclusive seat of the disease the general symptoms are to be mainly relied upon in making a diagnosis. The physical signs are indefinite and often altogether absent. Auscultation sometimes reveals feebleness of the vesicular murmur and a few subcrepitant rales at the base of the lung of the side affected. The attack begins suddenly, preceded by a chill and followed by high fever, violent pain, and excessive dyspnea. The pain is usually referred to the hypochondrium, extending to the epigastrium, and sometimes to the ileolumbar region of the corresponding side. It is increased by deep inspiration, and is made intolerable by hiccough and vomiting. The painful regions are very tender to the lightest touch. This is especially true, as has been pointed out by M. Gueneau de Mussy, of a spot one or two finger's breadth from the *linia alba* on the level with the bony part of the tenth rib. When this point is even slightly pressed upon the patient cries out, and there is also a sudden increase of dyspnea, seeming to threaten suffocation. This spot M. de Mussy called "*le bouton diaphragmatique*." The phenomenon is explained by a hypersensitive branch of the phrenic nerve. Pressing upon the hypochondrium from below upward also gives rise to great pain. The respirations are excessively frequent and almost convulsive in character. The patient breathes with the ribs rather than with the diaphragm. The difficulty of breathing is sometimes so great as to cause orthopnea, the patient leaning forward and supporting the painful part with the hand to immobilize the chest.

Hemorrhagic pleurisy is of rare occurrence in the primary form. In the majority of instances it is secondary to some organic disease or to a general infection. Its physical signs do not differ practically from those of the serofibrinous variety, and accurate information is only to be obtained by exploratory puncture.

Some years ago a man of middle age, who was under my care suffering from an acute exacerbation of chronic articular gout, was taken with pain in the right side, with rapid filling up of the pleural cavity. He had enlargement of the heart, with long-standing valvular disease and cirrhotic kidneys. On being tapped the pleural effusion was found to be hemorrhagic. Death occurred after a short illness from uremia.

Another patient under my care was a lad, aged eighteen years, of good previous health and about to enter college, who took ill with pyrexia which proved to be typhoid of irregular type. At the beginning of convalescence he was attacked with left-sided pleurisy,

followed by a rapid and copious hemorrhagic effusion. The patient after an illness of many weeks finally recovered sufficiently well to get about and retained improved health until several months later, when he was taken suddenly ill with tuberculous meningitis, ending fatally in about ten days.

Interlobar pleurisy with sacculated effusion, serous or purulent, is always difficult to diagnose, and often impossible to locate. It rarely exists alone, but is associated with extensive fibrinous or serofibrinous inflammation. A cystic empyema is the more common, and is sometimes an infection associated with pneumonia. Persistent fever, with increasing evening exacerbations and a high leukocyte count, leads to exploratory puncture, and if purulent fluid be found, operation is called for.

A colored woman, by occupation a cook, under my care a few years ago, was taken ill with pneumonia of the apex of the right lung, beginning with a violent chill and followed by fever, dyspnea, and cough, with rusty sputum, a pseudocrisis occurring on the ninth day of the patient's illness. The fever increased again with marked evening rise, lasting for many weeks. The leukocyte count varied from 17,000 to 24,000. The cough was most annoying, and the expectoration, scanty at first, increased in quantity and became purulent. Repeated exploratory punctures failed to reveal the presence of pus. The physical signs were localized dulness and rough breathing near the original seat of pneumonia, and impairment of resonance from the spine of the scapula to the base of the lung, with coarse friction and bronchovesicular respiration. After an exhausting illness of many months the patient was able to resume her former occupation. The case unquestionably was one of empyema in the fissure between the upper and middle lobe of the right lung, remaining undiscovered by puncture, and finally discharging through a bronchial fistula. Pleurisy and pneumonia are often combined; indeed, pneumonia rarely exists without some degree of plastic inflammation of the pulmonary pleura; but to properly constitute a case of pleuropneumonia there must be a decided effusion. More than the ordinary amount of pain indicates the coexistence of the two diseases, and if there be positive flatness on percussion at the base of the lung, lessened or absent vocal fremitus, indistinctness of bronchial breathing and perhaps egophony, the diagnosis can be no longer in doubt. The convalescence is more apt to end by lysis, and is prolonged by fluid remaining to be absorbed.

The physical signs of pleurisy vary according to the stage of the disease and the amount of effusion in the pleural cavity. Compared with the general symptoms they are of greater importance in forming a correct diagnosis. The expansion of the lower part of the affected side is less than that of the sound side, even before effusion occurs, mainly in consequence of painful breathing, and the cor-

responding side of the chest is also somewhat retracted as may be shown by measurement.

Palpation is not only of use in determining the degree of lessening of the respiratory movements, but is also of great importance in estimating the strength of the vocal vibrations, which are absent in deep effusions and feebly present in others, a physical sign of much importance from a diagnostic point of view. Palpation is a useful means of determining the degree of displacement of the heart and the extent of downward displacement of abdominal organs. In pleurisy with large effusion on the right side the free edge of the liver is often felt below the costal border, and if the left side be the seat of disease, the outlines of the spleen are sometimes distinctly made out.

The sounds on percussion in pleurisy are the most important of all of the physical signs. At the onset there is no material change, but when effusion occurs it gives rise to dulness on light percussion, becoming gradually flat as the liquid increases in quantity.

Fluid dulness has a character of its own, if percussion be not too strong, being airless without resonance, and different from the dulness of lung-consolidation, not unlike the sound obtained on striking the thigh. The flatness is more decided at the lower portion of the chest and becomes gradually less from below upward. If the disease be left-sided, allowance must be made for the resonance of the stomach, which often modifies the sound as far up as the eighth interspace. As the effusion increases the upper line of dulness when the patient is sitting upright is not horizontal but assumes more or less the shape of an extended letter S, curving upward and forward to the axillary region, thence downward to the lower part of the sternum. The percussion note above the level of a large effusion is dull and tympanitic, and in front below the clavicle it has high-pitched tympanitic or Skodaic resonance of exaggerated intensity, contrasting strongly with the flatness lower down. The depth of the sound and its tympanitic quality is one of the most characteristic signs of pleuritic effusion, extending up as high as the third or fourth rib anteriorly. When the pleural cavity is quite filled with fluid the percussion note is dull everywhere on the side affected, except at the root of the lung, where the bronchial tubes are large and rigid. Under these circumstances, if the disease be on the left side dulness on percussion replaces the normal tympanitic resonance of Traube's semilunar space following depression of the diaphragm, and with it the stomach which lies in close contact. In rare cases of large left-sided effusions the lifting power of the lung may be sufficient to counterbalance the weight of the fluid to the extent of limiting the depression of the diaphragm and not greatly interfering with the tympanitic resonance of Traube's space throughout the attack.

The auscultatory signs of pleurisy vary greatly according to the

extent of the inflammation, the amount of effusion, the degree of compression of the lung, and the number of adhesions. The respiratory murmur, somewhat enfeebled at first by restricted movement of the lung from pain, is still more so when liquid effusion occurs. As the effusion takes place the breath sounds become more feeble and rough, with prolonged expiration, which in the lower part of the chest may be altogether absent. Above the level of the fluid, where the compressed lung, still containing air, is in contact with the walls of the chest or only separated by a small amount of liquid, the respiration becomes more or less bronchial, its intensity varying according to the degree of condensation of the lung, and the voice is bronchophonic. Bronchial respiration and bronchophony coexist in pleurisy, but the latter has a vibration or quivering in its tone which never exists, at least to the same degree, in pneumonia proper. When the bronchial respiration is not loud the resonance of the voice becomes less bronchial, but its vibration is increased and so-called egophony is heard.

The sound of egophony is not of much diagnostic importance in pleurisy, because it requires for its production a thin stratum of liquid intervening between the lung and the side of the chest, and therefore a moderate pressure upon the bronchial tubes. The sound ceases entirely when the effusion is large or else it is converted into bronchophony, of which it is merely a modification. The sound has been known to be propagated through a layer of false membrane after the pleural liquid has been withdrawn. It has been called one of the fancy signs of pleurisy. Its full though rare development once heard is never forgotten. The egophonic voice, when it exists, is best heard between the anterior axillary line and the scapula or in the interscapular region of the affected side. It is more frequently heard in women and children because of the higher pitch of their voice.

Another sign of pleurisy more important than the resonance of the voice and pathognomonic of the disease is the friction sound. It is heard both upon inspiration and expiration, more especially the former, and is often irregular from sticking together of the opposed pleural surfaces, which are released by an unusually deep inspiration such as that after cough. The friction sound is most liable to be confounded with a rhoncus, which is itself often perceptible to the touch; but a friction sound is seldom so loud as a rhoncus, and is rather increased by deep inspiration, whereas a rhoncus is altered or ceases after a vigorous cough. It is also characteristic of a friction sound that it is rendered more distinct when firm pressure by the stethoscope or by the ear is made against the chest. Friction occurs under two different circumstances; at the beginning before serum is effused and toward the termination of the disease when the liquid has undergone absorption or has been evacuated by tapping and the two inflamed surfaces of the pleura again come

in contact. At the close of the disease it is coarser and louder, and is best heard below the angle of the scapula or in the lateral region of the thorax. One reason why a friction is so often overlooked is because it is a sign rather than a sound, as shown by slight jerking movements of the chest. The character of friction sounds vary from the finest crepitation to the sole-leather friction or "bruit de cuir neuf" sometimes heard at a distance from the patient and often perceptible to the touch. Pleural crepitation occurs in volley form, resembling the crepitant rale of pneumonia, but it is finer, more moist, and is directly under the ear. I have occasionally found it in association with the sounds of catarrhal bronchitis, especially when the patient had violent fits of coughing.

The physical signs of pleurisy in infancy and childhood show great variations and cannot alone be depended upon, but must be compared with the general symptoms and the history of the attack in making a diagnosis. Of the diseases of the chest in children, pleurisy is the disease in which mistakes in diagnosis are most often made. Fibroserous and suppurative pleurisy both occur in early life, but the purulent form is relatively more common in infancy and not infrequently occurs *d'emblée*. The prevalence of the purulent disease in infancy is shown by the modern records of the Hospital for Sick Children. Dr. Henry K. Dillard informed me once that when he was resident physician at the Children's Hospital in Philadelphia there were five infants, varying in age from ten months to two years, operated upon for empyema in one winter. The operation in each case was by free incision and resection of one rib. The patients all recovered. In the Children's House of the Bryn Mawr Hospital there were eleven patients, mostly infants, operated upon for empyema within one year. The physical signs of pleurisy in children often resemble those of pneumonia, and it requires great care on the part of the physician to avoid a diagnostic error. The chief source of confusion is the persistence of bronchial breathing throughout the dull region of a large effusion, with entire absence of vocal fremitus. In different parts of the affected side of the chest the respirations may be intensely tubular. Friction sounds are rarely heard in young children, and the same may be said of feebleness of the breath sounds. Another difference is the comparative absence of visceral displacement, which is accounted for by the flexible chest of the child yielding readily to fluid accumulation, pressure not being expended upon the adjacent organs to the same extent at least as in the adult.

The diagnosis of pleurisy is readily made in most instances. An uncomplicated and well-characterized case cannot be confounded with any other affection. That is, when distention of the chest, absolute dullness on percussion, faintness or absence of vocal fremitus, feebleness or absence of respiratory murmur, and displacement of

viscera coincide with pain, irregular breathing, and fever. If the general symptoms be too much relied upon the diagnosis in many cases would be puzzling, but with the aid of the physical signs a satisfactory conclusion can be promptly reached. Pneumonia, with pleurisy is often confounded, but differs from the latter in the violence of its initial chill and the character of the pain, which in uncomplicated pneumonia is moderate and dull. The cough of pneumonia is deeper and the sputum viscid and rusty. The chest is not enlarged, the vocal fremitus is often intensified in the dull region, and the heart and liver are not displaced. The dulness on percussion is not absolute as it is in pleurisy, and the resistance to the finger in the latter is greater. The semitympanitic resonance on percussion in pleurisy with large effusion characteristically exists above the level of the fluid. In rare cases a tympanitic sound on percussion is perceived in pneumonia, but it is then most pronounced over the consolidated lung. In pneumonia loud bronchial respiration is heard over the whole of the consolidated portion of the lung, but in pleurisy with large effusion, excepting in infancy and childhood, a tubular quality of breathing is rarely heard below the level of the fluid. There may be bronchial respiration in the dull area when a slight pleuritic effusion exists, but it will not be accompanied with bronchophony but with egophony, a state of voice rendered bleating by the interposition of a layer of fluid between the costal and pulmonary pleura.

The presence of pus in the pleural cavity is to be suspected when the temperature rises and assumes a persistent remittent form, the patient at the same time often complaining of a sense of chilliness. An auscultatory sign of value is the non-transmission to the ear of the whispered voice through the fluid (Bacilli) sign ("pectoriloque aphonique"). The only positive means of determining the character of the fluid is by exploratory puncture.

Hydrothorax is distinguished from pleurisy by the absence of fever in the former and by the fact that simple transudations are usually bilateral and are frequently associated with dropsy in other parts of the body. I have had the opportunity of observing three cases of unilateral serous transudation into the pleural cavity: one the result of circulatory pressure of a cancerous growth in the mediastinum and two from the pressure of enlarged bronchial glands in Hodgkin's disease.

Acute primary pleurisy in robust subjects usually ends in recovery without leaving serious after effects. But secondary pleurisies are always serious according to the gravity of the infection with which they are associated. In the treatment of the early stage, pain is much relieved by strapping the affected side of the chest or by the local application of a cold compress. When the patient dreads the cold, or if its application fails to keep the pain in check, hot flaxseed poultices, frequently changed may be substituted. An occasional

hypodermic injection of morphin gives additional comfort. The patient should be kept absolutely quiet in bed on restricted diet, until the decline of the fever. Internal medication is indicated by special symptoms. Diaphoretics are sometimes useful for modifying the pyrexia in the early stage of the disease. After the decline of the fever, the effusion having apparently reached its maximum, and when scanty secretion of the kidneys is still an existing symptom, the exhibition of diuretics is called for to stimulate absorption of the fluid. The bowels should also be kept freely evacuated, administering, if required, saline laxatives occasionally preceded by calomel followed by a purgative. The operation of thoracentesis should be performed in all cases in which the pleural cavity is largely filled with fluid, and in others with excessive dyspnea or when signs of absorption fail to appear. The internal and external use of iodine and finally of muriated tincture of iron will hasten absorption of fluid and inflammatory thickening of the pleuræ.

Pus in the pleural cavity should be promptly removed by free incision and rib resection to establish free drainage. An unexpanded lung and consequent retraction of one side of the chest are subject to improvement or cure by pulmonary gymnastics and systemitized exercise of the body in general.

RECURRENT PNEUMOTHORAX: REPORT OF A CASE, WITH REVIEW OF THE LITERATURE.

BY CLYDE L. CUMMER, M.D.,

CLEVELAND, OHIO.

MEDICAL literature is replete with studies of pneumothorax, and although cases of presumed spontaneous pneumothorax are not specially common, the subject has been well considered. In attempting to ascertain the frequency of recurrent pneumothorax we found that little had been written concerning it. In Albutt's *System*,¹ Finlay says that "as a curiosity attention may be directed to recurring pneumothorax: these cases are accompanied by few symptoms, nearly always recover, and may therefore be due to rupture of emphysematous bullæ." Neither in Osler's *Modern Medicine*² nor in Nothnagel's *Encyclopedia of Practical Medicine*³ is even mention made of this phase of the subject under the general head of "pneumothorax."

In 1888 Gabb⁴ reported a case which he had seen in one attack

¹ Albutt and Rolleston, 1909, v, 575.

² Walter R. James, iii, 868.

³ O. Rosenbach, *Pneumothorax*, p. 972.

⁴ Recurrent Pneumothorax, *British Med. Jour.*, 1888, ii, 178.